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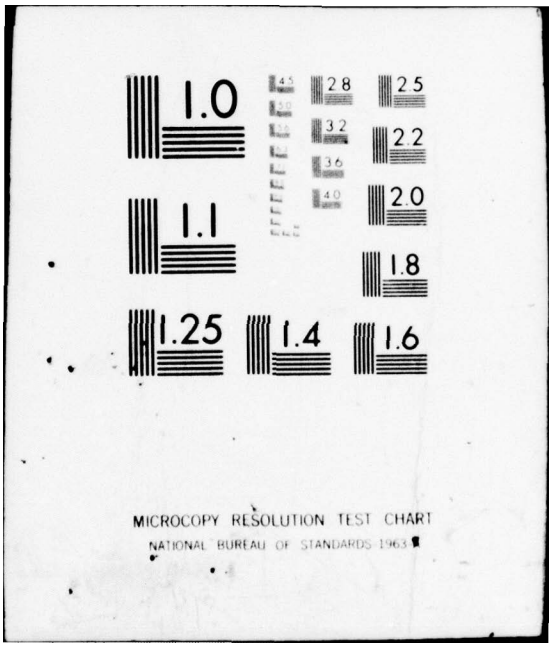
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TECHNICAL REPORT NO. II

PROTOCOL FOR MANAGEMENT OF PENETRATING CAROTID ARTERY INJURY

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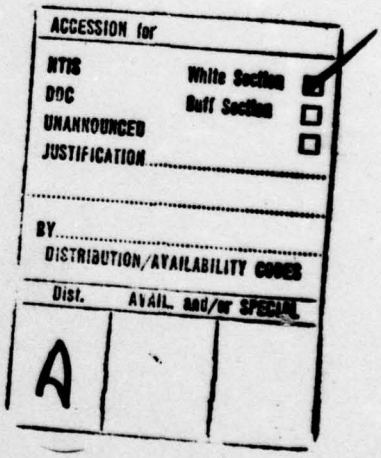

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Following the conflict in Southeast Asia a number of civilian and military reports were published in an effort to establish guidelines for management of carotid arterial injuries.[2,3,4,8,13,16,17] These articles focused on the preoperative neurological state of the patient and attempted to establish guidelines for repair based on the presence or absence of neurologic deficits. The conclusions were vague, however, leaving the surgeon with confusing criteria on which to base his treatment. This report reviews a 10 year experience at the Medical College of Virginia of isolated common and internal carotid injuries in eighteen patients. Comparison with published reports of 215 cases has allowed us to develop specific clinical guidelines for management of carotid arterial injuries.

PATIENTS AND METHODS

From January 1966 to December 1975, 96 patients with penetrating injuries to the neck underwent surgical exploration at the Medical College of Virginia, Virginia Commonwealth University. Eighteen of them had isolated injuries to the common or internal carotid arteries. Those patients with associated injuries that might have influenced their clinical course were eliminated from review, as were those with isolated external carotid or venous injuries.

The delay from time of injury to surgical intervention could be determined accurately in 12 cases and the mean interval was three hours and fifteen minutes. Seventeen of the eighteen patients were male and the age range was from 19 to 70 years with a mean of 31 years. There was an equal distribution of right and left sided injuries. Sixteen wounds were inflicted by low caliber gunshot wounds and there were two penetrating injuries from knife wounds.

A thorough neurological evaluation was performed preoperatively in each patient. The comatose state was defined as unresponsiveness to verbal stimuli. Systemic hypotension defined as systolic blood pressure less than 90 mmHg was

present on admission in seven patients and was corrected preoperatively in each case. There was no correlation between hypotension, neurological deficit and the end result (Tables 1 and 2). Four patients had significant neurological deficit and associated hypotension. Correction of hypotension did not improve the neurological deficit.

There were four deaths (mortality rate 22%), all occurring in the immediate post-operative period (i.e., within 72 hours). All deaths were related to cerebral injury. Post-mortem examination was obtained in one of the patients and showed macroscopic evidence of hemorrhagic infarction. The second patient who died had a clinical course compatible with uncal herniation. The third death followed 12 hours of generalized seizure activity. Subsequent EEG's showed no evidence of cerebral activity. In the fourth patient, disseminated intravascular coagulation complicated the clinical picture, although the neurological status of the patient was unchanged following surgery.

ABSENCE OF PRE-OPERATIVE NEUROLOGIC DEFICIT:

On examining the preoperative neurological status of the patients, ten of them showed no deficit (Table 1). All had primary vascular repair of their arterial injury and nine of them are now alive and well without neurological sequelae. One patient (Case #5) had a transient monoparesis which cleared completely. Another patient (Case #4) developed a left hemiparesis post-operatively. This patient had a benign course until the fifth post-operative day when he developed a left hemiplegia and rapidly became comatose. He underwent emergency cranial decompression by right frontal lobectomy. The pathology report showed hemorrhagic infarction of the right frontal lobe. At two years follow-up, this patient is ambulatory with a brace and is under treatment for a seizure disorder.

PRE-OPERATIVE NEUROLOGICAL DEFICIT:

In eight cases (Table 2), the patient had a significant neurological deficit

after hypotension had been corrected. The deficits consisted of monoplegia, hemiplegia or quadriplegia, with or without an associated comatose state. All deaths occurred in this group.

Four patients had operative repair of their vascular injury. Patients #12 and #14 had localized deficits manifested as monoplegia and hemiplegia, respectively. Both of them cleared their deficit following revascularization. Patients #13 and #17 were comatose and patient #13 was also quadiplegic. Both of them died within 72 hours following revascularization.

An additional four patients were managed by ligation of the carotid system. All of these patients were comatose at the time of surgery and three of them also had localizing neurological signs. There were two deaths in the group, one of which occurred nine months following surgery. One patient has shown improvement, and the fourth is fully recovered.

COLLECTED REVIEW:

A number of reports related to carotid arterial injury have been published since 1963.[1,2,3,4,6,8,13,16,17] The preoperative and postoperative neurological status and operative management were determined in each series. Those cases with associated head injury or any significant injury which could influence the outcome of the patient were eliminated. A total of two hundred fifteen cases was obtained through this search. Combined with the cases from this study a total of two hundred thirty-three cases were included in the following analysis.

The cases were divided into four groups, cross matching the presence or absence of preoperative neurological deficit with the method of management; i.e., restoration of vascular continuity or ligation of the damaged carotid vessel.

One hundred and seventy patients had a normal neurological examination prior to surgery. One hundred and sixty-one of them had direct repair of their injury. There were no deaths in this group but two patients developed hemiplegia as a residual neurological deficit[16] (Table 3).

Nine patients who had normal neurological examinations had the injured carotid vessel ligated. This was usually due to technical difficulty associated with attempts at repair. There was one death in this group and two patients developed permanent neurological deficits[4,13] (Table 3).

Sixty-three patients had abnormal neurological examinations preoperatively. The deficits were monoplegia, hemiplegia, quadriplegia, aphasia, or coma. The comatose state was often found in conjunction with lateralizing signs. Forty-nine patients had restoration of vascular continuity. There were twelve deaths in this group and all of them were related to cerebral injury. The overall results are shown in Table 4.

The remaining fourteen patients were managed by ligation of the carotid artery (Table 4). Forty-three percent were no worse following surgery but fifty-seven percent deteriorated or died following carotid artery ligation.

If those patients who were comatose at the time of surgery are excluded, then the results following vascular repair as opposed to ligation are exceptionally good (Table 5). There were thirty-four patients who had significant preoperative neurological deficits (monoplegia, hemiplegia, aphasia) but without coma. Twenty-nine of them (85%) were improved or showed no deterioration from their preoperative state. Twenty-two of them (65%) had a complete recovery with subsequent normal neurological examination. There was progression of the neurological deficit in two patients and three of them died.[4] It should be noted that in these three deaths no mention of the presence or absence of coma was made in the preoperative assessment. It is assumed that no coma was present; therefore, any bias would be directed against the final conclusions of this study. The fate of the comatose individual is bleak. Seventy percent died regardless of the mode of therapy (Table 6).

DISCUSSION:

The current surgical literature including the most recent reports from Southeast Asia contains a number of pleas for more conservative approaches to management of patients with penetrating carotid injuries in association with severe neurological deficits.[2,4] Bradley, in an excellent review[2] which included a pathophysiological study of injury based largely on the primate experience of Denny-Brown and Meyer,[5,9-12] proposed that any injury with neurological lesions more severe than mild paresis, should be managed by ligation. Cohen[4] advocated the same approach following his military experience, except that he favored ligation in every patient with any degree of neurological impairment. Thal[16] recommended vascular repair only if flow was present in the carotid system at the time of exploration. The recent experience at the Medical College of Virginia and the review of published data do not support these conservative recommendations.

It is logical to assume that the presence of a preoperative neurological deficit is an indication of inadequate cerebral collateral blood flow. However, this assumes that those factors which influence collateral blood flow are corrected before determining the extent of the deficit. These factors include systemic hypotension, reduction in arterial oxygen tension and accumulation of acid metabolites.[9] On this basis, patients with penetrating carotid injuries can be divided into two groups: A. Those patients with adequate collateral flow as evidenced by the absence of neurologic signs. B. Those patients with inadequate collateral circulation as demonstrated by the presence of a neurological deficit. The exception to this would be those patients with injury to the carotid system without total disruption of flow. In this group, the adequacy of collateral flow cannot be assessed.

Ninety percent of the cerebral blood flow is contributed by the carotid vessels; ten percent by the vertebral system. If flow is interrupted in one of

these vessels, there is a compensatory increase in flow in the other vessels supplying the circle of Willis.[7] However, only twenty percent of normal individuals have what is considered to be the standard anatomical configuration.[14] Other collateral pathways exist, but their importance in the acute disruption of flow is unknown.[2]

It seems apparent from the clinical results that the neurological state which correlates best with an irreversible injury is coma. Motor weakness or paralysis may be reversible with a very acceptable risk factor. Improvement therefore may be dependent on re-establishing blood flow to the ischemic area. Many believe that there is an area of relative ischemia surrounding an infarcted region which, although physiologically inert, can improve with an increase in or restoration of cerebral blood flow. There is experimental evidence that permanent interruption by ligation of a major vessel decreases the reserve capacity of the brain to tolerate any vascular stress.[15] Consequently, experimental evidence and the clinical data presented argue against carotid ligation.

The management of the comatose patient remains difficult. Results are similar regardless of which surgical approach is taken. It seems reasonable that if prograde flow is present in the injured carotid at the time of exploration, then revascularization should be attempted. If no flow is present and the patient is comatose, restoration of flow to this avascular area could be deleterious.

This study has focused on isolated carotid injuries in an effort to delete any bias that associated injuries might have on the end result. Our experience with patients having carotid injury in association with multiple system involvement is small, but the extension of this treatment plan to the multiple injured patient appears logical. The carotid injury must take high priority in the immediate management of these individuals.

CONCLUSIONS:

I. All patients who do not have a neurological deficit should have restoration of vascular continuity.

This method of treatment is associated with uniformly good results, does not subject the patient to permanent interruption of flow and the possible sequelae, and does not place the patient at greater risk due to the progression of atherosclerotic disease.

II. All patients who have a significant neurological deficit (monoplegia, hemiplegia, quadriplegia, or aphasia) short of coma, should also have primary vascular repair. This method of treatment has been shown to be significantly superior to carotid artery ligation.

III. In comatose patients, repair or reconstitution should be undertaken if prograde flow is present. Neither repair nor ligation appear to change the basic poor prognosis significantly.

IV. At the present time, ligation of the carotid artery is indicated only:

(a) for the comatose patient with no prograde flow.

(b) for technical reasons when repair is impossible.

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Table 1. Results of Surgical Repair of Carotid Artery Injuries in Patients without Preoperative Neurological Deficits at the Medical College of Virginia 1966-1976

Sex	Age	Mode of Injury	Injury[a]	Assoc Injury	Operation	Preoperative Neuro Status	Postoperative Neuro Status	Shunt	Comment
M	19	GSW	C.C.	None	Resection & Primary Anastomosis	Normal	No Deficit	Yes	Normotensive: ischemic time 4 minutes
M	70	GSW	C.C.	None	Lateral Arteriorrhaphy	Normal	No deficit	Yes	Normotensive
F	58	GSW	C.C.	None	Resection & Primary Anastomosis	Normal	No deficit	No	Normotensive: ischemic time 12 minutes
M	24	GSW	C.C.	Left Linear Skull	Resection & Primary Anastomosis	Normal	L. Hemiparesis	No	Hypotension corrected preoperatively; developed hemiparesis on 5th post-op day. Emergency right frontal lobectomy
M	20	GSW	C.C.	None	Saphenous Vein Graft *CC to IC*	Normal	Transient Paresis, right arm	Yes	Normotensive: cleared completely
M	50	Stab	C.C.	None	Resection & Primary Anastomosis	Normal	No deficit	No	Hypotension corrected preoperatively
M	32	GSW	C.C.	None	Ext. Jugular Vein Patch C.C.	Normal	No deficit	Yes	Normotensive
M	35	GSW	C.C.	A-V Fistula	Resection & Primary Anastomosis	Normal	No deficit	Yes	Normotensive
M	24	GSW	I.C.	None	Lateral Arteriorrhaphy	Normal	No deficit	No	Normotensive
M	20	Stab	C.C.	None	Dacron Patch Graft to C.C.	Normal	No deficit	No	Hypotension corrected preoperatively. Respiratory arrest necessitating tracheostomy in E.R.

[a] CC = Common carotid artery
IC = Internal carotid artery

Table 2. Results of Surgical Repair of Carotid Artery Injuries in Patients with Preoperative Neurological Deficits at the Medical College of Virginia 1966-1976

Sex	Age	Mode of Injury	Injury[a]	Assoc Injury	Operation	Preoperative Neuro Status	Postoperative Neuro Status	Shunt	Comment
M	20	GSW	C.C.	Fracture Mandible	Ligation Common Carotid Bifurcation	Unresponsive sluggish pupils	No deficit	Yes	Normotension corrected preoperatively. Ischemic time 10 minutes
M	21	GSW	C.C.	None	Resection & Primary Anastomosis	R. monoplegia (arm)	Deficit Cleared	Yes	Normotensive
M	19	GSW	C.C.	Transected Spinal Cord @ C-5 level	Resection & Primary Anastomosis	Unresponsive Flaccid extremities	Death at 72 hrs.	Yes	Hypotension corrected in O.R. prior to surgery
M	26	GSW	C.C.	None	Saphenous Vein Graft C.C. to I.C.	Left hemiparesis	Deficit Cleared	Yes	Normotensive
M	46	GSW	C.C.	None	Ligation Common Carotid	Comatose with right hemiparesis	Death	No	Normotensive - Generalized seizure activity postoperatively. Death at 72 hours
M	21	GSW	C.C. I.C.	None	Resection & Primary C.C. to I.C.	Comatose	Decerebration & Death		Autopsy confirmed intracerebral hemorrhage
M	20	GSW	C.C.	Int. jug vein, soft tissue	Ligation Right Common Carotid	Comatose	Comatose		Hypotension corrected preoperatively: DIC
M	30	GSW	C.C.	C.C.	Ligation Common Carotid	Comatose left hemiplegia	Unchanged		Hypotension corrected preoperatively. Death 2° aspiration 9 months postoperatively. Remained comatose

[a]CC = Common carotid artery
IC = Internal carotid artery

Table 3. Collected Results of the Treatment of Carotid Artery Injuries in the Absence of Preoperative Neurological Deficit

Treatment	Revascularized	Ligated
Previously Published Cases	151	9
Current Report	10	0
Total Cases	161	9
Results		
No Deficit	153	6
Transient Deficit	6	0
Permanent Deficit	2	2
Death	0	1
Favorable Outcome	99%*	67%*

*$p < 0.05$

Table 4. Collected Results of Treatment of Carotid Artery Injuries in the Presence of Preoperative Neurolgical Deficit

Treatment	Revascularized	Ligated
Previously Published Cases	45	10
Current Report	4	4
Total Cases	49	14
Results		
Improved	23	5
Unchanged	10	1
Progressive Deficit	4	1
Death	12	7
Favorable Outcome	67%*	43%*

*Not Significant

Table 5. Collected Results of Treatment of Carotid Artery Injuries in the Presence of Preoperative Neurological Deficit Exclusive of Coma

Total Cases Reported - 40

	Revascularized	Ligated
Improved	22	2
Unchanged	7	1
Progressive Deficit	2	1
Death	3	2
Favorable Outcome	85%*	50%*

*$p < 0.05$

Table 6. Collected Results of Treatment of Carotid Artery Injuries in the Presence of Coma

Total Cases Reported - 23

	Revascularized	Ligated
Improved	4	2
Unchanged	0	0
Progressive Deficit	2	0
Death	9	6
Favorable Outcome	27%*	25%*

*Not Significant

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Results of treatment of patients with penetrating isolated carotid artery injuries with or without neurological damage have been reviewed at the Medical College of Virginia and compared with published experience from the surgical literature. On the basis of these outcomes a Protocol For Management is described, emphasizing the value of revascularization surgery in all cases including coma when prograde flow is present. Ligation of the carotid artery following injury may still be indicated for technical reasons and in comatose →

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